Acid-base balance

« اللهم انفعني بما علمتني وعلمني ماينفعني وزدني علمًا »

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Acid-base balance

- The data are used to assess patients in life-threatening situations
- Blood hydrogen ion concentration [H+] is maintained within tight limits in health. Normal levels lie between 36 and 44 nmol/L (pH 7.35-7.45)

arterial ← → venous.

- Any H+ values outside this range will cause alteration in the rates of chemical reactions within the cell and affect many metabolic processes of the body

* أي خلل جاد PH الدم يأثر على شغل enzymes و pumps وكل metabolic process بتضرر.

- Values greater than 120 nmol/L or less than 20 nmol/L are usually incompatible with life

≃ (6.90 ≤ PH) (7.70 ≥ PH)

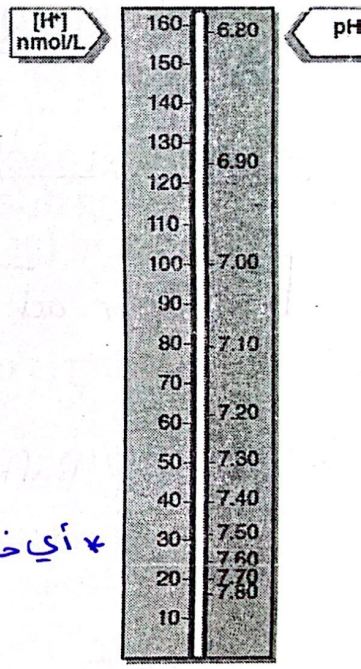


Fig 20.1 **The negative logarithmic relationship between [H+] and pH.**

H+ Production

* جاي من شغلتين :-
1. metabolism
2. CO_2 retention in body

- Hydrogen ions are produced in the body as a result of **metabolism** (from the oxidation of the sulphur-containing amino acids of protein ingested as food)

- The total amount of H+ produced each day in this way is of the order of 60 mmol but all the H+ produced are efficiently excreted in urine. Everyone who eats a diet rich in animal protein passes an acidic urine

* كل H+ اللي بناكله بيطلع من الجسم على شكل acidicurine .

- Large amounts of **CO2** are produced by cellular activity each day with the potential to upset acid-base balance

- Under normal circumstances all of this CO2 is excreted via the **lungs**. Having been transported in the blood, Only when **respiratory function** is impaired do problems occur

* CO_2 الناتج من الخلايا بيطلع عن طريق air exchange داخل lungs
لـ في حال عنا مشكلة بالرئتين بيرجع CO_2 للدم وما بيطلع من lungs .

Buffering and buffers

- Buffer is a solution of the salt of a weak acid that is able to bind H+. Buffering does not remove H+ from the body but mop up any excess H+ produced (as a sponge)
  - Buffer act on small quality of pH change لـ (بتشتغل زي الإسفنجة بتمسح شغلة صغيرة)

- **Buffering** is only a short term solution to the problem of excess H. Ultimately, body must get rid of H by renal excretion
  - لما يكون كمية H^+ كبيرة بدي تشتغل H^+ زيادة (كتير) بعين Buffer ما بيكفي .

- The body contains a number of buffers to correct sudden changes in H production

- **Proteins** can act as buffers and the haemoglobin in the erythrocytes has a high capacity to bind H

* لما الشخص يصير عنده alkalosis/acidosis بتحاول كل محاليل Buffers تعدلها ما زبطت .

Buffers

$HCO_3^- + H^+ \rightleftharpoons H_2CO_3 \rightleftharpoons CO_2 + H_2O$

- In the ECF, **bicarbonate buffer** is the most important. In this buffer system, bicarbonate (HCO3-) combines with H+ to form carbonic acid (H2CO3)
- The association of H with bicarbonate occurs rapidly, but the breakdown of carbonic acid to CO2 and water happens relatively slowly.
- The reaction is accelerated by an enzyme, **carbonic anhydrase**, which is present particularly in the erythrocytes and in the kidneys.
- Only when all the bicarbonate is used up does the system have no further buffering capacity
- The acid base status of patients is assessed by consideration of the bicarbonate system in plasma

Buffers

- The **bicarbonate buffer** system is unique in that:
  - The (H2CO3) can dissociate to water and CO2 allowing CO2 to be eliminated by lung
  - Changes in CO2 modify the ventilation rate
  - HCO3- concentration can be altered by the kidney
- **Phosphate buffer** system (HPO4= - H2PO4-) plays a role in plasma and RBC's and is involved in the exchange of Na/H+ ion in the urine filtrate
- **Plasma proteins**, especially the imidazole groups of histidine, forms important buffer system in plasma. Most circulating proteins has net negative charge capable of H+ binding

Regulation of the acid-base balance

- In plasms at 37oC, the value for the combination of the solubility constant for PCO2 and the factor to convert mm Hg to mmol/L is 0.0307 mmol L-1. mm Hg-1

$$pH = pK' + \log \frac{cHCO_3^-}{0.031 \times PCO_2}$$

7.35 or 7.45 ← pH بتعطيني اياها — $cHCO_3^-$ بتعطيني اياها — PCO_2 بتعطيني اياها

* بحسب قديش بدي HCO_3^- عشان أحدل pH للدم (normal pH)

= معنى سؤال امتحان .

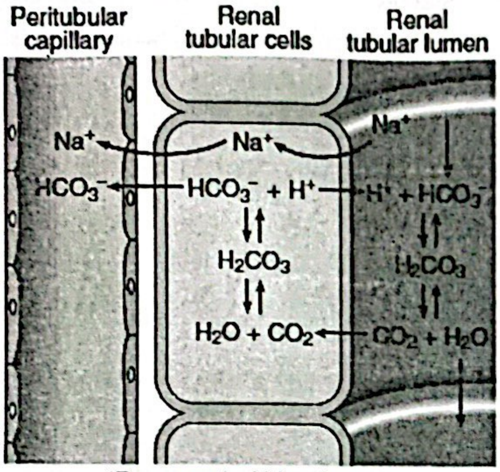
H+ excretion in the kidney

- All the H+ that is buffered must eventually be excreted from the body via the kidneys, regenerating the bicarbonate used up in the buffering process and maintaining the plasma bicarbonate concentration within normal limits.
- Secretion of H+ by the tubular cells serve initially to reclaim bicarbonate from the glomerular filtrate so that it is not lost from the body
- When all bicarbonate has been recovered, any deficit due to the buffering process is regenerated.
- The mechanisms for bicarbonate recovery and for bicarbonate regeneration are very similar and sometimes confused.
- The excreted H+ must be buffered in urine or the [H+] would rise to very high levels, phosphate acts as one such buffer, while ammonia is another

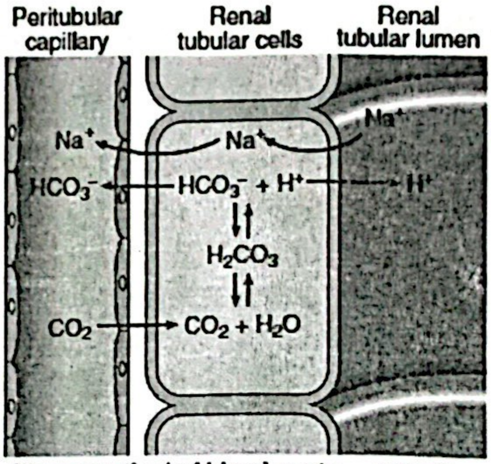
* كل HCO_3^- اللي بيطلع بال urine بيرجع بيصيرله اعادة امتصاص للدم .

H+ excretion in the kidney

(Blood) (urine) (Blood) (urine)



'Recovery' of bicarbonate



'Regeneration' of bicarbonate — excretion of hydrogen ion → لا يكون شي مشكلة. Respiratory (in respiratory acidosis)

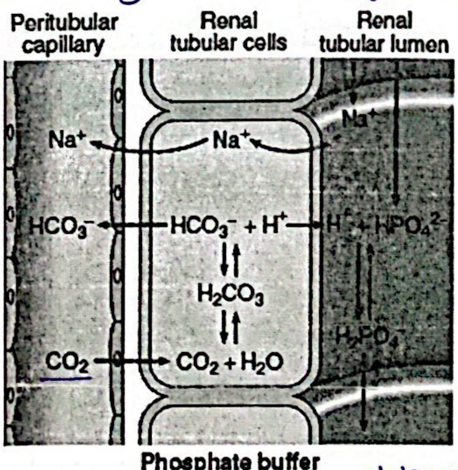
* بتخلص من H^+ بربطها ح HCO_3^- وبتطلع على شكل H_2CO_3.
* بتتفكك بال lumen ل H^+ + HCO_3^- بيطلع H^+ مع urine وبترجع HCO_3^- للدم.
* بدخل Na^+ بدل H^+.

* بطلع CO_2 من الكلية بتحول ل H^+ + HCO_3^-.
* H^+ بيطلع من الجسم مع urine وبصير إعادة امتصاص ل HCO_3^-.

← هيك يكون تخلصت من CO_2 وزدت HCO_3^- بالدم فيترفع PH.

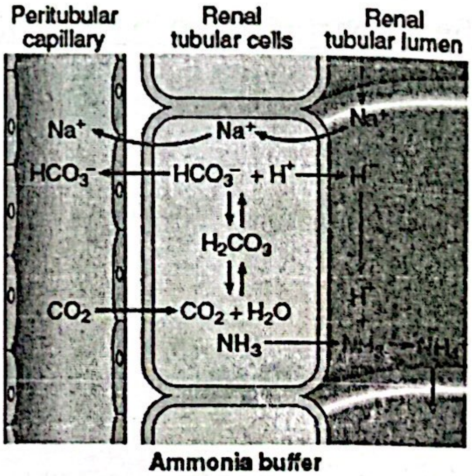
H+ excretion in the kidney

= هدف Buffer بيعادلو H^+ داخل urine عشان high acidity بتحرق urinary track.



Phosphate buffer

= بيرتبط H^+ بال urine مع HPO_4^{-2}.



Ammonia buffer

= بيرتبط H^+ مع NH_3 بال urine.

* ممكن سبب alkalosis / acidosis او metabolic او respiratory .

Assessing status

• اذا كان الخلل metabolic بيجدله Respiratory system ⇐ سريع
• اذا كان الخلل Respiratory بيجدله renal system ⇐ بطيء .

- The carbonic acid (H_2CO_3) component is proportional to carbon dioxide, which is in turn proportional to the partial pressure of the CO_2
- Because the body's cellular and metabolic activities are pH dependent, the body tries to restore acid-base homeostasis whenever an imbalance occurs (**Compensation**)
- The body accomplishes this by altering the factor not primarily affected by the pathologic process. For example, if the imbalance is of **non-respiratory** origin, the body compensates by altering **ventilation** (fast response).

- For disturbances of the **respiratory** components. The **kidneys compensate** by selectively excreting or reabsorbing anions and cations. The kidneys are slower to respond (2-4 days)

Assessing status

- The H concentration in blood varies as the bicarbonate concentration and pCO_2 change. If everything else remains constant.

- Adding H (1), removing bicarbonate (2) or increasing the pCO_2 (3) will all increase $[H^+]$ (acidosis)

- Removing H, adding bicarbonate or lowering pCO_2 will all cause the $[H^+]$ to fall. (alkalosis) hyperventilation

- An indication of the acid-base status of the patient can be obtained by measuring the components of the bicarbonate buffer system

Normal ranges

TABLE 16-1 ARTERIAL BLOOD GAS REFERENCE RANGE AT 37°C

pH	7.35–7.45
pCO_2 (mm Hg)	35–45
HCO_3^- (mmol/L)	22–26
Total CO_2 content (mmol/L)	23–27
pO_2 (mmol/L)	80–110
SO_2 (%)	>95
O_2Hb (%)	>95

Causes of metabolic acidosis

- Metabolic acidosis with an **elevated anion gap** occurs in:

* كل اشي برجع retained

- **Renal disease.** Hydrogen ions are retained along with anions such as sulphate and phosphate.

- **Diabetic ketoacidosis.** Altered metabolism of fatty acids, as a consequence of lack of insulin causes endogenous production of acetoacetic and β–hydroxybutyric acids

acidosis + gap بكل ketonbodies

- **Lactic acidosis.** Particularly tissue anoxia. In acute hypoxic states such as respiratory failure or cardiac arrest. It can be caused by liver disease. The presence of lactic acidosis can be confirmed by the measurement of plasma lactate concentration.

Lactate المسؤول عن التخلص من liver

- Certain disease of overdosage or **poisoning.** As in **salicylate** overdose where build-up of lactate occurs, or **methanol** poisoning when formate accumulates, or **ethylene glycol** poisoning where oxalate is formed.

Salicylate ⇒ lactate

methanol → Formaldehyde

ethylene glycole ⇒ oxalate.

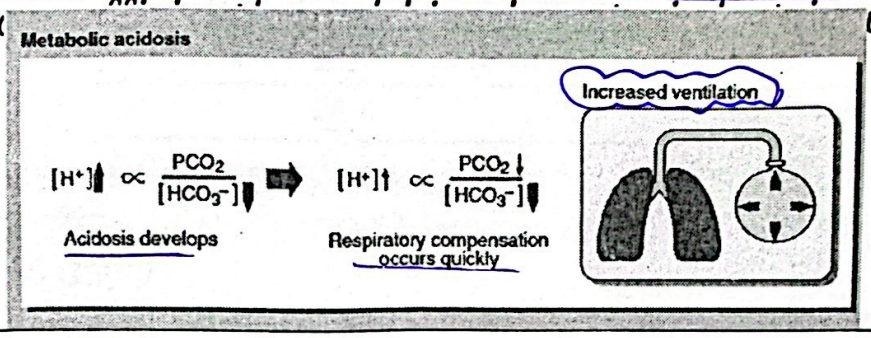
Causes of metabolic acidosis

- Metabolic acidosis with a normal anion gap is sometimes referred to as **hyperchloraemic acidosis** because a reduced HCO_3 is balanced by increased Cl concentration. It is seen in chronic diarrhea or intestinal fistula. Fluids containing bicarbonate are lost from the body

لما عشان الجسم بيفقد HCO_3^- بحاول يعوض بال Cl^- فبيصير عند الشخص Hyperchloraemia.

- **Renal tubular acidosis**: Renal tubular cells are unable to excrete hydrogen i... urine

H^+



Clinical effect of acidosis

- The compensatory response to metabolic acidosis is hyperventilation, since the **increased [H+]** acts as a powerful stimulant of the respiratory centre.

- The deep rapid and gasping respiratory pattern is known as Kussmaul breathing. Hyperventilation is the appropriate physiological response to acidosis and it occurs rapidly.

- The raised [H+] leads to increased neuromuscular irritability. There is a hazard of arrhythmia progressing to cardiac arrest and this is more likely by the presence of hyperkalemia, which will accompany the acidosis.

- Depression of consciousness can progress to coma and death

Respiratory acidosis (pH لتعويض Renal system بشتغل الحالة هاي)

د أسباب Respiratory acidosis ع

- **Lung disease:** in which CO2 is not effectively removed from the blood. In certain patients with chronic obstructive pulmonary disease (COPD, where CO2 is retained in the blood, causing chronic hypercarbia (elevated pCO2)

- In **bronchopneumonia:** gas exchange is impaired because of the secretions. White blood cells, bacteria and fibrin in the alveoli

- **Hypoventilation** caused by drugs such barbiturates (1), morphine (2), or alcohol (3) will increase blood pCO2 levels (أسباب ٣)

- **Mechanical obstruction** or asphyxiation (strangulation or aspiration).

- **Decreases cardiac output** such as in CHF also will result in less blood to the lungs for gas exchange and an elevated pCO2

- Kidney will compensate for acidosis but it takes time

Respiratory alkalosis (pH لتعويض Renal System عمان هون بشتغل)

- The causes include:
  1. Hypoxemia ⇒ (حالدم) → Hypoxia (all over the body)
  2. Chemical stimulation of the respiratory center by drugs, such as salicylate
  3. An increase in environmental temperature, fever, hysteria (hyperventilation), Pulmonary emboli and pulmonary fibrosis.

- The kidney compensates by excreting HCO3- in the urine and reclaiming H+ to the blood
- The popular treatment for hysterical hyperventilation, breathing into a paper bag, is self -explanatory

= يتنفس المريض دبكيس من ورق ، عشان ازيد كمية CO_2 داخل الـ lungs.